WHAT IS CLAIMED IS:

1. An isolated σ^{54} polypeptide comprising an amino acid sequence set forth in SEQ ID NO:2, and conservative variants thereof.

2. A polynucleotide selected from the group consisting of:
   (a) SEQ ID NO:1;
   (b) SEQ ID NO:1, wherein T is U;
   (c) nucleic acid sequences complementary to (a) or (b); and
   (d) fragments of (a), (b), or (c) that are at least 15 nucleotide bases in length and that hybridize to DNA under highly stringent conditions including 0.1 x SSC at about 68°C for 15 minutes which encodes the polypeptide set forth in SEQ ID NO:2.

3. An isolated polypeptide comprising an amino acid sequence encoded by a nucleic acid sequence which hybridizes to the nucleic acid sequence set forth in SEQ ID NO:1 under highly stringent conditions, wherein the highly stringent conditions include 0.1 x SSC at about 68°C for 15 minutes, and wherein the polypeptide is a transcription factor.

4. The polypeptide of claim 3, wherein the polypeptide binds to a LuxO protein.

5. An isolated polypeptide that binds to an antibody generated against the polypeptide of SEQ ID NO:2, or fragments and conservative variants thereof, wherein the polypeptide is a transcription factor.

6. A vector containing the polynucleotide of claim 2.

7. A host cell containing the vector of claim 6.

8. An anti-polypeptide antibody that binds to the polypeptide of claim 3.

9. A method for regulating the activity of a σ^{54} polypeptide comprising contacting σ^{54} with a LuxO polypeptide.

10. The method of claim 9, wherein the contacting is *in vivo*.

11. The method of claim 9, wherein the contacting is *in vitro*.

12. The method of claim 9, wherein the σ^{54} polypeptide is from *V. harveyi*.

13. A method for identifying a compound that regulates the binding of a LuxO polypeptide to a σ^{54} polypeptide comprising:

(a) contacting a σ^{54} polypeptide with a LuxO polypeptide under conditions and for such time as to allow binding of the σ^{54} polypeptide to the LuxO polypeptide;

(b) contacting the σ^{54} polypeptide or LuxO polypeptide of (a) with the compound prior to, simultaneously with, or after binding of the σ^{54} polypeptide to the LuxO polypeptide; and

(c) measuring the binding of the σ^{54} polypeptide to the LuxO polypeptide in the presence of the compound and comparing it to the binding of the LuxO polypeptide with the σ^{54} polypeptide in the absence of the compound,

wherein, a change in the binding of a LuxO polypeptide to a σ^{54} polypeptide in the presence of the compound is indicative of a compound that regulates LuxO-σ^{54} binding.

14. The method of claim 13, further comprising manufacturing the compound so identified.

15. The method of claim 13, further comprising formulating the compound so identified with a pharmaceutically acceptable carrier.

16. The method of claim 13, wherein the contacting is *in vivo*.

17. The method of claim 13, wherein the contacting is *in vitro*.

18. The method of claim 13, wherein the modulation is by inhibition of LuxO-σ^{54} binding.

19. The method of claim 13, wherein the compound is a polypeptide.

20. The method of claim 13, wherein the compound is a small molecule.

21. The method of claim 13, wherein the compound is a LuxO analog.

22. The method of claim 13, wherein the compound is a σ^{54} analog.

23. The method of claim 13, wherein the compound is a nucleic acid.

24. A pharmaceutical composition comprising a compound identified by the method of claim 13 in a pharmaceutically acceptable carrier.

25. The pharmaceutical composition of claim 24 in a controlled release formulation.

26. The pharmaceutical composition of claim 25 in a liposomal form.

27. The pharmaceutical composition of claim 24 in a lyophilized form.

28. The pharmaceutical composition of claim 23 in a unit dosage form.

29. A method for identifying a compound that inhibits LuxO-σ^{54} binding comprising:

(a) contacting a mixture comprising LuxO and σ^{54} with the compound under conditions and for such time as to allow LuxO-σ^{54} binding;

(b) contacting (a) with a bacterial cell, or extract thereof, comprising biosynthetic pathways which will produce a detectable amount of light in response to LuxO-σ^{54} binding; and

(c) measuring the effect of the compound on light production,

wherein decreased light production in the presence of the compound, compared to light production in the absence of the compound, identifies the compound as a compound that inhibits LuxO-σ^{54} binding.

30. The method of claim 29, wherein the compound is a competitive inhibitor.

31. The method of claim 29, wherein the compound is a suicide inhibitor.

32. A method for identifying a compound that regulates the activity of a LuxO-σ^{54} complex, comprising:

(a) contacting a LuxO-σ^{54} complex with the compound; and

(b) measuring the activity of the complex in the presence of the compound and comparing the activity of the complex obtained in the presence of the compound to the activity of the complex obtained in the absence of the compound;

wherein a change in the activity of the LuxO-σ^{54} complex in the presence of the compound is indicative of a compound that regulates LuxO-σ^{54} complex activity.

33. A method for regulating expression of a virulence factor in a bacterial cell comprising contacting a bacterial cell capable of producing the virulence factor with a compound identified by the method of claim 13, claim 29 or claim 32.

34. The method of claim 33, wherein the regulating is inhibition of expression of the virulence factor.

35. The method of claim 33, wherein the virulence factor is a siderophore.

36. The method of claim 33, wherein the virulence factor is selected from the group consisting of Accessory cholera enterotoxin, Adenylate cyclase toxin, Adhesin, Aerolysin toxin, aggregation substance, Agr A,B,C,D, SigB etc, Alkaline protease, Alpha toxin, Alpha-haemolysin, Alveolysin, Anthrax toxin, APX toxin, Beta toxin, Botulinum toxin, Bundle forming pilus structural subunit, C2 toxin, C3 toxin, C5A peptidase, Cardiotoxin, Chemotaxis, Cholera toxin, Ciliotoxin, Clostridial cytotoxin, Clostridial neutotoxin, Collagen adhesion gene, Crystal endotoxin, CyaA toxin, Cytolysin, Delta toxin, Delta toxin, Delta-lysin, Diphtheria toxin, Emetic toxin, Endotoxin, Staphylococcal Enterotoxins A, B, C1, C2, C3, D, E, G , Enterotoxin, Exfoliative toxin, Exotoxin, Exotoxin A, Exotoxin B, Exotoxin C, Extracellular elastase, Fibrinogen, Fibronectin binding protein i.e. fnbA, Filamentous hemagglutinin, Fimbriae, Gamma hemolysin, Gelatinase i.e.gelE, Haemolysin, Hemolysin B, Hemagglutinin, Histolyticolysin, IGG binding protein A i.e. spaI, Intimin, Invasin, Iron siderophores, Ivanolysin, Ivanolysin O, Lantibiotic modifying enzyme, Lantibiotic structural protein, Lecithinase, Ler (positive regulator of LEE genes), Leukotoxin, Lipoprotein signal peptidase, Listeriolysin O, M protein, Motility, Neurotoxin, Nonfimbrial adhesins, Oedema factor, Perfringolysin O, Permease, Pertussis toxin, Phospholipase, Pili, Plasmid encoded regulator per, Pneumolysin, Poly-D-glutamic acid capsule, Pore-forming toxin, Proline permease, RNAIII, RTX toxin, Serine protease, Shiga toxinm, Siderophore/iron acquisition protein, SigA proteases, Spe A, Spe B, Spe C, STa toxin, Stb toxin, Streptolysin O, Streptolysin S, Superantigen, Superoxide dismutase, TCP, Tetanus toxin, Thiol-activated cytolysin, Tracheal cytotoxin, TSST toxin (TSST-1), and Urease, Zona occludens toxin.

37. The method of claim 33, wherein the bacterial cell is a pathogenic bacterial cell.

38. The method of claim 33, wherein the bacterial cell is selected from the group consisting of *Vibrio harveyi, Vibrio cholerae, Vibrio parahaemolyticus, Vibrio alginolyticus, Pseudomonas phosphoreum, Yersinia enterocolitica, Escherichia coli, Salmonella typhimurium, Haemophilus influenzae, Helicobacter pylori, Bacillus subtilis, Borrelia burgfdorferi, Neisseria meningitidis, Neisseria gonorrhoeae, Yersinia pestis, Campylobacter jejuni, Deinococcus radiodurans, Mycobacterium tuberculosis, Enterococcus faecalis, Streptococcus pneumoniae, Streptococcus pyogenes and Staphylococcus aureus*.

39. A method for regulating expression of a siderophore polypeptide in a bacterial cell comprising contacting a bacterial cell capable of producing the siderophore polypeptide with a compound identified by the method of claim 12, claim 26 or claim 29.

40. The method of claim 39, wherein the regulating is inhibition of expression of the siderphore polypeptide.

41. The method of claim 39, wherein the bacterial cell is a pathogenic bacterial cell.

42. The method of claim 39, wherein the bacterial cell is selected from the group consisting of *Vibrio harveyi, Vibrio cholerae, Vibrio parahaemolyticus, Vibrio alginolyticus, Pseudomonas phosphoreum, Yersinia enterocolitica, Escherichia coli, Salmonella typhimurium, Haemophilus influenzae, Helicobacter pylori, Bacillus subtilis, Borrelia burgfdorferi, Neisseria meningitidis, Neisseria gonorrhoeae, Yersinia pestis, Campylobacter jejuni, Deinococcus radiodurans, Mycobacterium tuberculosis, Enterococcus faecalis, Streptococcus pneumoniae, Streptococcus pyogenes and Staphylococcus aureus*.

43. A method for regulating bacterial colony morphology comprising contacting a bacterial colony with a compound identified by the method of claim 13, claim 29 or claim 32.

44. The method of claim 43, wherein the regulating is inhibition of smooth colony morphology formation.

45. The method of claim 43, wherein the bacterial colony contains pathogenic bacterial cells.

46. The method of claim 45, wherein the bacterial cells are selected from the group consisting of *Vibrio harveyi, Vibrio cholerae, Vibrio parahaemolyticus, Vibrio alginolyticus, Pseudomonas phosphoreum, Yersinia enterocolitica, Escherichia coli, Salmonella typhimurium, Haemophilus influenzae, Helicobacter pylori, Bacillus subtilis, Borrelia burgfdorferi, Neisseria meningitidis, Neisseria gonorrhoeae, Yersinia pestis, Campylobacter jejuni, Deinococcus radiodurans, Mycobacterium tuberculosis, Enterococcus faecalis, Streptococcus pneumoniae, Streptococcus pyogenes and Staphylococcus aureus.*

47. A method for treating a subject infected with a pathogenic bacterial cell comprising administering to the subject an inhibitor or antagonist that regulates LuxO binding to σ^{54}.

48. A method for inhibiting bacterial cell growth or virulence in a subject, comprising administering to the subject an inhibitor or antagonist that regulates LuxO binding to σ^{54}.

49. The method of claim 48, further comprising killing the bacterial cell after inhibiting its growth.

50. The method of claim 49, wherein the bacterial cell is killed by administering an antibiotic agent.

51. The method of claim 49, wherein the bacterial cell is killed by action of the immune system of the patient.

52. A bacterial cell comprising a distinct alteration in the rpoN gene, wherein the alteration results in an rpoN$^-$ phenotype.

53. The cell of claim 52, wherein the bacterial cell is *Vibrio harveyi rpoN*::Cm^r.